# Foreign Animal Disease Report

United States Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services
Emergency Programs



Number 12-4 December 1984

In This Issue



## Current Events

Avian Influenza

The Federal area quarantines in Pennsylvania and Virginia for lethal avian influenza, reported in the last three issues (12-1, 12-2, 12-3), have been released. At the time this issue went to press, quarantines remained on three premises in Pennsylvania because cleaning and disinfecting (C&D) had not been completed or the required 30-day period following C&D had not elapsed.

The Virginia quarantine was released September 14, 1984, after a total of 1,249,264 birds comprising 65 poultry flocks from which the causative virus was isolated, had been depopulated. A total of 19,392 birds in 4 seropositive flocks were also depopulated. Four other flocks were depopulated before the eradication program began. The last lethal avian influenza virus in Virginia was isolated June 27, 1984.

The Pennsylvania quarantine was released October 4, 1984, after 15,754,439 birds comprising 379 flocks were depopulated. Flocks depopulated had either lethal avian influenza virus, low pathogenic virus of the same H5N2 serotype, or specific antibodies.

A total of 716 farms in Virginia and 839 farms in Pennsylvania were under surveillance for avian influenza on October 15. Active surveillance will be continued in both previously quarantined areas for 6 months. Surveillance will include investigation of any flock reported sick or suspicious, and laboratory testing of regularly collected egg yolk, blood serum, and dead birds. (Dr. K. A. Hand, 301 436-8065)

ASF Update Haiti

On September 3, 1984, the Haitian Minister of Agriculture declared Haiti had met the Office International des Epizooties (OIE) standard for declaring Haiti free of African swine fever (ASF). The last positive ASF serology was disclosed March 3, 1984, in a feral pig from the La Pierre area. No ASF virus was isolated from that pig. Intensive hunting has been

conducted in the La Pierre area. No pigs have been found in that area since July 1984.

Recently, visits were made to 523 sites where 1,408 sentinel swine are kept. Approximately 100 sentinel sites remain to be visited. Total sentinel sites increased from 502 to approximately 620 primarily due to peasants redistributing sows to places where feed is available.

Twenty-two sentinel pigs died during the last 9 months. All were free of ASF.

All sentinels will be tested for serum antibodies to ASF virus in the near future. (Dr. James A. Downard, 317 248-4132)

Screwworm Update Mexico

The Mexico-U.S. Screwworm Eradication Program entered the final months of the eradication phase in mid-1984 (see Screwworm Program Review, June issue 1983). By December 31, 1984, the program objective will change from insect eradication in additional areas, to the maintenance of a sterile fly barrier at the Isthmus of Tehuantepec. This should prevent reinfestation of the screwworm-free areas of Mexico and the United States. Only nine occurrences of screwworms were reported north of the barrier zone during the summer of 1984. All of these resulted from animal shipments from the States of Chiapas and Tabasco. These States are in southern Mexico where eradication efforts are continuing.

Insecticide treatment by dipping or spraying is being enforced by the military at three quarantine stations. This should greatly lower the number of new cases resulting from animal movements, thereby reducing the likelihood of reinfestation. (Mr. W. H. Sudlow, 301 436-5986)

World Animal Disease Roundup

The summer and early fall months went by with little news about exotic diseases. Whether this was due to lack of reporting or lack of diseases may have to be left for speculation. There was only one report of foot-and-mouth disease (FMD) from Africa (Zimbabwe, type $SAT_3$). The disease appeared again in West Germany: type O on a single pig farm in Bavaria. No new cases were reported in Greece. In the Americas, cases were reported in Argentina, Brazil, Ecuador, Paraguay, and Colombia. No further cases were found in Chile. Elsewhere we hear that the disease was seen in Malaysia, Hong Kong, Laos, Israel, and Lebanon.

Rinderpest was reported only from Kuwait, but of course it still exists in Africa, where preparations for a massive eradication campaign continue.

Contagious bovine pleuropneumonia was reported in Namibia, where it is considered endemic. Spain reported a case in June. This is of special interest because of its possible link with outbreaks reported earlier in Portugal and France.

Reports on African swine fever came from Italy, Portugal, Cameroon, and Malawi.

Hog cholera is still being reported from most swine-raising areas in the world. West Germany led the list of affected countries, reporting almost 100 cases per month.
(Dr. H. J. Seyffert, 301 436-8285)

**Manila Office Opens**

Increasing interests and commitments worldwide are making it difficult for APHIS Veterinary Services to keep abreast of all necessary details directly from headquarters in Washington, DC. For many years, an APHIS veterinarian posted at the U.S. Embassy in Rome has represented the agency in Europe, Africa, and the Near East. Dr. Claude Nelson is the current incumbent. His responsibilities include the collection of animal disease information, liaison with foreign government officials and international organizations active in the animal health field, and facilitating U.S. exports of animals and animal products. Similar responsibilities are now being met for the Far East and Pacific areas by APHIS veterinarian Dr. James Cavanaugh, who is posted at the U.S. Embassy in Manila, Philippines. Plans are underway to establish additional representation for the Caribbean Basin, South America, and Africa.
(Dr. H. J. Seyffert, 301 436-8285)

**Malignant Catarrhal Fever**

Malignant catarrhal fever (MCF) is a generalized viral disease of cattle and many species of wild ruminants characterized by high fever, corneal opacity, panophthalmitis, generalized lymphadenopathy, leukopenia, and in most cases, profuse nasal discharge and necrotic inflammation in the oral and nasal cavities, sometimes extending into the esophagus and trachea.

In cattle, MCF has been recognized for centuries in Africa by both African pastoral tribesmen and early settlers. They associated its occurrences in cattle with areas inhabited by wildebeest (Connochaetes spp.), especially during the wildebeest calving season. In other areas of the world, MCF in cattle has been associated since the late 1700s with contact with sheep, especially during lambing.

MCF Viruses and Hosts

The causative virus of wildebeest-associated MCF was first isolated by Plowright in Kenya in 1960 from a blue wildebeest (Connochaetes taurinus taurinus). He characterized this viral isolate as a highly cell-associated herpesvirus. More recent studies suggest that this virus should be classified as a member of the subfamily Gammaherpesvirinae, of the family Herpesviridae, because of characteristics it shares with other gamma herpesviruses, such as Marek's disease virus, Epstein-Barr virus, Herpesvirus saimiri, H. ateles, and H. sylvilagi. These characteristics include high lymphotropism with associated lymphoid cell proliferation, asymptomatic latent virus carriers, virus strongly cell associated, absence of inclusion bodies in vivo, but development of typical Cowdry Type A inclusion bodies and syncytia in cell cultures, and highly variable incubation periods. The name alcelaphine herpesvirus-1 has been proposed for MCF herpesviruses isolated from wildebeest, and alcelaphine herpesvirus-2 for those isolated from hartebeest (Alcelaphus buselaphus). These species are members of the subfamily Alcelaphinae, of the family Bovidae.

The virus of sheep-associated MCF has not yet been isolated. The clinical and pathologic manifestations of MCF of sheep or alcelaphine antelope origin are essentially indistinguishable.

MCF Serology

Recent serologic evidence suggests that the agent of sheep-associated MCF is related to alcelaphine herpesviruses. Antibodies which react strongly with alcelaphine herpesviruses by indirect immunofluorescence and weakly or not at all by virus neutralization, have been found in sera of domestic and wild sheep and goats. In wild sheep and goats, the prevalence of such antibodies has been between 25 and 30 percent. Similarly reactive antibodies have been found in the sera of domestic cattle with non-wildebeest-associated MCF. The prevalence of such antibodies in U.S. cattle remains to be determined.

MCF Epidemiology

The occurrence of several focal epidemics of MCF in cattle and captive wild ruminants in zoos in the United States, Europe, and Asia, and on commercial deer farms in New Zealand prompted a serologic survey for MCF antibodies in captive exotic ruminants in U.S. zoos. A high prevalence of MCF virus neutralizing antibodies and indirect immunofluorescent antibodies was found in several species. These included all species of wildebeest (Connochaetes spp.), hartebeest (Alcelaphus spp.) and topi (Dalmaliscus spp.). All are members of the subfamily Alcelaphinae. Other species with high prevalence of MCF antibody include scimitar-horned oryx (Oryx gazella dammah), fringe-eared oryx (Oryx gazella callotis), gemsbok (Oryx gazella gazella), and addax (Addax nasomaculatus), which are members of the subfamily Hippotraginae. Species in the

subfamily Caprinae, which include wild and domestic sheep and goats, in which a high prevalence of MCF antibody exists, are: Cretan wild goat (Capra aegagrus cretica), Turkomen markhor (Capra falconeri heptneri), African pygmy goat (Capra aegagrus hircus), and European mouflon sheep (Ovis orientalis musimon). Recently, MCF virus neutralizing antibodies were found in serum from a cottontail brush rabbit at the San Diego Wild Animal Park, an endemic focus of MCF.

The alcelaphine herpesvirus of MCF has been isolated in the United States from asymptomatic, seropositive white-tailed gnu (wildebeest), white-bearded gnu, Cape hartebeest, topi, and scimitar-horned oryx. During the past 5 years, similar viruses have been isolated in U.S. zoos from greater kudu, Indian gaur, nilgai, Formosan sika deer, axis deer, barasingha deer, and Watusi (Ankole) cattle with clinical MCF.

The recent growth of exotic game ranching as a commercial enterprise, and as a way to conserve rare endangered hoofed animals, has coincided with an apparent increase of clinical MCF in both captive exotic ruminants and domestic cattle. This suggests a need to increase diagnostic and surveillance activities for MCF to assess the possible impact of this disease on livestock and game ranching.

Shedding of cell-free MCF virus has been demonstrated in nasal and ocular secretions and feces of neonatal wildebeest calves up to 4 months of age. Cell-free alcelaphine herpesvirus has also been isolated from nasal secretions of stressed, corticosteroid-treated adult wildebeest, and from nasal and ocular secretions of Formosan sika deer with acute clinical MCF. MCF virus has been found to be entirely cell-associated in nasal secretions of cattle afflicted with alcelaphine MCF. Circumstantial evidence suggests that MCF-affected cattle sometimes transmit MCF to other cattle by contact. The circumstances permitting this to occur need further study.

MCF Diagnosis

A preliminary diagnosis of MCF is based upon clinical signs, gross pathology and, especially, microscopic pathology. Typical microscopic changes include disseminated vasculitis and lymphoreticular proliferation in lymph nodes. Some cases may be confused with bluetongue, bovine viral diarrhea, and vesicular diseases.

Definitive diagnosis is based upon the demonstration of MCF virus in blood leukocytes or other tissues and demonstration of the appearance or increase in titer of MCF virus neutralizing antibodies in the serum of affected animals, or both. Tissues most suitable for virus isolation include whole unclotted blood, lymph nodes, tonsils, spleen, adrenal gland, liver, lung, and kidney. These should be submitted to the laboratory chilled but not frozen. Blood should be collected in an anticoagulant: EDTA (ethylenediamine tetraacetate), or heparin, or ACD (USP formula A anticoagulant containing sodium citrate, citric acid, and dextrose). In the laboratory, leucocytes from the blood sample are cultivated or fused with susceptible cell cultures of

bovine or ovine kidney, lung, spleen, thyroid, or adrenal gland. This culture method has been proved the most successful for alcelaphine MCF virus isolation. (For further details, see Heuschele, W. P. and Castro, A. E., Malignant Catarrhal Fever. In Foreign Animal Diseases, Their Prevention, Diagnosis, and Control, pp. 244-255, U.S. Animal Health Assn. Richmond, Virginia, 1984.)

Isolation of sheep-associated MCF virus has been unsuccessful by in vitro methods. However, rabbits have been successfully infected by inoculating them with blood and tissues from affected cattle and deer. Lymphoid cell lines from MCF-infected rabbits have been established and have transmitted MCF by inoculation and passage in laboratory rabbits. The rabbit-passage virus has not been characterized.

MCF Prevention

Prevention of MCF requires that there be no contact between reservoir or carrier species, and susceptible cattle or other ruminant species, particularly those of Asian origin.

A safe efficacious vaccine for MCF is not available.

MCF Risk

MCF appears to be an emerging disease with the potential of becoming a hazard for the cattle industry and the ranching and propagation of endangered exotic ruminants in captivity. Serologic testing of exotic ruminants may be necessary to assure their freedom from MCF before they are allowed to enter the United States. The exclusion of seropositive animals should reduce the potential for spread by carriers into uninfected herds and currently unaffected areas. (Dr. Werner P. Heuschele, Zoological Society of San Diego, San Diego, CA 92112)

FMD in Elephants

The following review summarizes available literature on a subject of special interest. References from which this information was taken are available in the Emergency Programs Information Center (EPIC) Data Bank (AHP, VS, APHIS, USDA, Hyattsville, Maryland 20782).

Foot-and-mouth disease (FMD) in elephants has been described as an acute, usually transient febrile disease, producing temperatures as high as 40.5°C to 41°C, loss of appetite, lameness, vesicles in the mouth and skin around the toenails, and accumulation of fluid in the feet resulting in separation and loss of part or all of the foot pad. Swelling of the ventral aspect of the trunk has also been reported in Indian elephants that were naturally infected with type O FMD virus.

Vesicles appeared at the injection sites in the tongues of African elephants 24 hours after they were given type SAT2 FMD virus. The vesicles ruptured at 72 hours. Lameness began at the 5th or 6th day of infection. Raised, blanched areas appeared around the toenails 2 to 5 days later, and healing began 1 or 2 weeks after the loss of epithelium. Healing was accompanied by deformities of the feet that were clearly visible for at least 10 months. Even though high concentrations of virus were present in blood, affected tissues, and vesicular

fluid, virus was not found in probang specimens of oesophageal-pharyngeal material collected at the 38th, 62nd, and 91st days of the study. Virus-neutralizing antibodies reached maximum titers of 1:64 at the 21st day and declined to as low as 1:6 by the 91st day. Uninoculated control animals kept with the infected ones remained normal and did not develop antibodies. This failure to spread by contact is in contrast to another instance in which type O FMD virus infected 16 of 30 Indian elephants that were brought together for a coronation ceremony in Nepal in 1975.

Relatively few reports of FMD in elephants have appeared in the literature. The available data support a conclusion that elephants infected with FMD virus may develop high viral titers and have an uncertain potential to spread the virus by contact with susceptible animals. (Dr. E. I. Pilchard, 301 436-8087)

FADR in Spanish

The Foreign Animal Disease Report (Noticias Sobre Enfermedades Exoticas de los Animales) is published in the Spanish language by the Comision Mexico-Americana para la Prevencion de la Fiebre Aftosa. Persons who wish to receive copies should contact Dr. John Mason, Assistant Co-Director, Aftosa Commission, American Embassy, P.O. Box 3087, Laredo, Texas 78041.

## Focus on... Hog Cholera

Hog Cholera (HC) is an acute, chronic, or inapparent highly contagious disease of swine. Although HC has been shown to replicate in many species, such as the rabbit which was used to produce a vaccine strain of HC virus, the only natural reservoir of HC is swine.

In an attempt to provide uniform terminology, the Commission of European Communities has designated the disease as classical swine fever in English, Schweinepest in German, peste porcine classique in French, and peste suina in Italian. However, in the Systematized Nomenclature of Veterinary Medicine, SNOVET (American Veterinary Medical Assn., 1984), it is named hog cholera.

History

Hog Cholera was first recognized in Franklin, Tennessee, in 1810, and is thought to have originated in the United States. The 1810 report of cholera-like disease preceded acute outbreaks of the disease in Indiana and Ohio in the early 1830's. Massive economic losses were being attributed to HC by 1856. The lack of an adequate grain marketing system in the 1800's caused farmers to increase swine production in order to dispose of surplus corn. Swine were also used to dispose of distillery waste. A distillery in Richmond, Indiana, lost 11,000 hogs in the summer and fall of 1856. In 1857, the American Veterinary Journal reported the loss of over 60,000 hogs within a 100-mile radius of Cincinatti due to a "destructive disease." The Bureau of Animal Industry (BAI) estimated that 13 percent of the U.S. swine population was killed by HC in 1886.

Hog Cholera continued to spread unchecked until after 1906 when Marion Dorset and coworkers in the BAI proved that simultaneous vaccination with hyperimmune serum and virulent HC virus produced long-lasting protection. Swine producers then had a means to prevent devastating losses due to HC and maintain production. By 1927 over 1 billion ml of HC hyperimmune serum was produced, and annual production remained over 1 billion ml until safer modified live virus (MLV) vaccines were introduced in the 1950's. Application of these vaccines reduced the incidence of virulent HC. By 1956, over 90 percent of vaccines used in the U.S. were MLV. Due to the marked efficacy of MLV vaccines and absence of side effects, industry pressure developed for removal of virulent virus/serum vaccination. By 1959 most of the major swine-producing States prohibited the use of virulent virus. Although apparent clinical disease was reduced, MLV vaccines caused losses due to abortion, stillbirths, and the shaker pig syndrome. Low virulent field strains of HC virus emerged and caused chronic production losses in baby pigs that failed to grow at a normal rate due to postnatal infection (Stewart, Diseases of Swine, Fifth Ed., Iowa State Univ. Press, 1981). Other problems also occurred with MLV vaccines, related to their degree of attenuation. Less attenuated MLV vaccines, if not administered with hyperimmune serum, actually caused chronic HC.

Continued losses led to the initiation of a State-Federal HC eradication program in 1962. During the eradication program, a peak number of 1,481 herds was reported as infected in 1969 (Wise, HC and Its Eradication, USDA, 1981). The eradication program progressed steadily thereafter, and the last case of HC occurred in New Jersey on August 1, 1976. The United States was declared free of HC January 31, 1978. Total cost of the eradication effort amounted to about $140 million in State and Federal funds.

## Geographic Distribution

Worldwide, HC remains epizootic. Only three countries in the Western Hemisphere are presently recognized by the United States as free of the disease: Canada, the United States, and the Dominican Republic. In the Eastern Hemisphere, Australia, Denmark, Finland, Great Britain, Iceland, New Zealand, Northern Ireland, the Republic of Ireland, Sweden, and the Trust Territory of Pacific Islands are recognized as free of HC.

The countries listed by the Office International des Epizooties (OIE) as having HC are shown in tables 1 and 2. Several countries are listed with no reported HC or HC vaccine, but they do not meet the requirements of the United States for recognition as free of HC. To be recognized free of HC, the country must 1) declare itself free of HC; 2) have a veterinary service that is capable of rapid detection of HC; 3) have veterinarians in its service that are graduates of a recognized school of veterinary medicine and have them assigned in sufficient numbers with respect to the swine population to be able to promptly recognize the existence of HC; 4) have access to a laboratory that is capable of diagnosing HC; 5) have mandatory reporting of HC to veterinary authorities of the

country; 6) have laws and regulations in effect to insure against the importation of swine, swine meat, and swine products from countries where HC exists; and 7) have quarantine station facilities and require quarantine and laboratory tests prior to the importation of swine into the country from HC-infected countries.

**Transmission**

The major means of transmission of HC is contact of susceptible swine with infected pigs. Virus is shed in urine, feces, and nasolacrimal secretions. In affected countries introduction of newly purchased swine into herds, particularly from dispersal sales, livestock shows, and sale barns, has frequently been shown to be a source of new HC infections.

Garbage feeding is a significant source for the introduction of HC into previously unaffected areas. Uncooked or improperly cooked pork scraps from infected swine may contain massive amounts of HC virus.

The inapparently infected carrier sow is another means of introducing HC virus into susceptible herds. This is a major mechanism of spread of HC in countries of the European Economic Community (EEC). When a pregnant sow is exposed to a low virulent field strain of HC or is inoculated with MLV vaccine, the baby pigs become infected in utero and develop a persistent infection (Stewart, Diseases of Swine, Fifth Ed. Iowa State Univ. Press, 1981). Termination of the pregnancy may occur as abortion, stillbirth, or both. Some infected pigs may be born alive. Infection with other disease agents such as leptospira, pseudorabies virus, parvovirus, and enterovirus may also cause abortion and stillbirth. In some cases, carrier sows have farrowed normal-appearing pigs with persistent viremias. These continuously excreted virus and later developed clinical disease. Affected pigs died as late as 11 months after birth. Van Oirschot and Terpstra (Vet Microbiol 2:121, 1977) reported that clinical signs in such pigs were not observed before 9 to 28 weeks of age--average 20 weeks--and the average survival time after clinical signs appeared was 6 months--range 2 to 11 months.

Table 1--Hog Cholera in the Western Hemisphere - 1983*

| Country | Outbreaks | Vaccination | Doses |
|---|---|---|---|
| Antigua & Barbuda | None | No | |
| Argentina | 16 | Yes | 1,900,000 |
| Bahamas | None | No | |
| Barbados | None since 1973 | No | |
| Belize | Moderate occurrence | Yes | NA |
| Bermuda | None | No | |
| Bolivia | Moderate occurrence | Yes | NA |
| Brazil | 140 | Yes | 6,200,000 |
| Chile | 20 | Yes | 800,000 |
| Colombia | 40 | Yes | NA |
| Costa Rica | No cases | NR | |
| Cuba | None since 1974 | Yes | 3,765,300 |
| Ecuador | Moderate occurrence | Yes | NA |
| El Salvador | Moderate occurrence | Yes | NA |
| Grenada | None reported | NR | |
| Guatemala | High occurrence | Yes | NA |
| Guyana | No cases | No | |
| Haiti** | Sporadic in feral swine | No | |
| Honduras | 40 | Yes | est. 50,000 |
| Jamaica | None | No | |
| Mexico | 484 | Yes | 13,506,000 |
| Nicaragua | High occurrence | Yes | NA |
| Panama | None since 1959 | No | |
| Paraguay | 5 | Yes | 85,000 |
| Peru | 9 | Yes | |
| St. Lucia | None since 1973 | No | |
| St. Vincent & & Grenadines | None | No | |
| Surinam | No cases | No | |
| Trinidad & Tabago | None since 1974 | No | |
| Uruguay | None since 1978 | Yes | 120,000 |
| Venezuela | 8 | Yes | 564,000 |

*Information derived from the Animal Health Yearbook published by FAO, WHO, and OIE for 1983, and Zoosanitary Situation in Member Countries in 1983 published by OIE.

** Hog cholera not reported in Haiti since March 3, 1984

NA - Not Available
NR - Not Reported

Table 2--Hog Cholera in the Eastern Hemisphere - 1983*

| Country | Outbreaks | Vaccination | Total Doses |
|---|---|---|---|
| Albania | None since 1973 | Yes | NA |
| Austria | 3 | No | |
| Belgium | 26 | Yes | 6,000,000 |
| Bhutan | Moderate occurrence | Yes | NA |
| Bulgaria | None reported | Yes | NA |
| Burma | Moderate occurrence | No | |
| China | Sporadic occurrence | No | |
| Cyprus | None since 1967 | No | |
| Czechoslovakia | None reported | Yes | 10,672,000 |
| F.R. Germany | 508 | No | |
| France | 13 | No | |
| French Polynesia | None reported | No | |
| German D.R. | Sporadic occurrence | Yes | NA |
| Greece | 1,416 | Yes | NA |
| Guinea-Bissau | Sporadic occurrence | NR | |
| Hong Kong | Sporadic occurrence | Yes | NA |
| Hungary | None since 1972 | No | |
| India | 29 | Yes | NA |
| Israel | None since 1959 | No | |
| Italy | 48 | Yes | 8,500,00 |
| Japan | 8 | Yes | 15,850,000 |
| Kampuchea | Moderate occurrence | Yes | NA |
| Laos | High occurrence | No | |
| Lebanon | Sporadic occurrence | No | |
| Luxembourg | 1 | No | |
| Macao | Sporadic occurrence | No | |
| Madagascar | Sporadic occurrence | Yes | NA |
| Malaysia | | | |
| Peninsula | 32 | Yes | NA |
| Sarawak | None reported | No | |
| Malta | None since 1968 | No | |
| Namibia | None since 1917 | No | |
| Nepal | High occurrence | Yes | NA |
| Netherlands | 161 | Yes | NA |
| Norway | None since 1963 | No | |
| Papua, New Guinea | None reported | No | |
| Philippines | Moderate occurrence | Yes | NA |
| Poland | None since 1978 | Yes | NA |
| Portugal | Moderate occurrence | Yes | NA |
| Republic of South Africa | None since 1918 | No | |
| Reunion | None reported | Yes | NA |
| Romania | None since 1974 | Yes | NA |
| Samoa | None reported | No | |
| Singapore | Sporadic occurrence | Yes | NA |
| South Korea | 3,436 | Yes | 3,900,000 |
| Spain | 10 | Yes | 4,311,682 |
| Switzerland | None since 1975 | No | |
| Taiwan | 155 | Yes | 7,275,000 |

Table 2--Continued

| Country | Outbreaks | Vaccination | Total Doses |
|---|---|---|---|
| Thailand | Sporadic occurrence | Yes | 917,291 |
| USSR | None reported | Yes | 72,970,000 |
| Vietnam | 166 | Yes | 78.75 percent of pig population |
| Yugoslavia | None since 1980 | Yes | 8,322,762 |

*Information derived from the Animal Health Yearbook published by FAO, WHO, and OIE for 1983, and Zoosanitary Situation in Member Countries in 1983, published by OIE.
NA - Not Available
NR - Not Reported

Neonatal pigs can also provide a reservoir of HC virus when they develop a persistent viremia. Experimentally infected pigs have had a persistent viremia for up to 121 days. Pigs infected with HC virus by Baker and Sheffy (Proc Soc Exp Biol Med 105:675, 1960) failed to grow but survived as long as 6 to 17 weeks. Mengeling and Cheville (Proc 72nd Ann Mtg US Lvstk Sanit Assoc, p 283, 1968) produced chronic HC in 22 of 69 pigs inoculated with an isolate from an Iowa epizootic. Sixteen of the pigs survived for more than 30 days.

In endemic areas where vaccination is used to control clinical HC, use of less attenuated MLV vaccines has resulted in contact spread of vaccine virus to baby pigs. These pigs subsequently developed clinical disease with varying mortality rates.

Mechanical transmission may also be a significant means for the spread of HC virus. Spread has followed the movement of contaminated personnel, farm equipment, pets, and wildlife. Trucks used to transport susceptible swine from a sale shortly after hauling pigs that were shedding HC virus have been linked to herd infection. Feed and rendering trucks moving between premises have also been incriminated for spreading HC virus. Mechanical spread by insect vectors, include biting flies and mosquitoes, has been reported.

Pathogenesis

Major consideration should be given to the less apparent forms of HC since these may not be readily recognized by producers or practitioners. Less apparent HC often results in unthrifty pigs without a major increase in the mortality rates expected in baby pigs and feeder pigs. This type of disease can be produced by less attenuated MLV vaccines, when administered without hyperimmune serum, and by low virulent field strains. Experimentally infected pigs develop a low grade fever and become anorexic and depressed. After several weeks the appetite and general appearance of the pigs improves markedly and their body temperatures return to normal or slightly above normal. Leukopenia, typical of acute HC, persists despite apparent recovery. Eventually the pigs relapse and die (Mengeling and

Cheville, Proc 72nd Ann Mtg US Lvstk Sanit Assoc, p. 283, 1968). The several weeks that have elapsed allow time for spread of infection from the affected herd.

Some less attenuated MLV strains may produce only an increased mortality in baby pigs with little or no disease in associated adult swine and feeder pigs. A rapid, accurate differential diagnosis is then critical to distinguish HC from pseudorabies and other septicemic diseases of baby pigs.

The affinity of HC virus for cells of the reticuloendothelial system results in the development of characteristic lesions. This virus also has an affinity for epithelial cells, but without associated lesions. The virus ordinarily enters the pig through ingestion, by aerosol, or by direct contact with the mucous membranes, conjunctiva, or skin abrasions. The primary replication site is the tonsil, with secondary spread to regional lymph nodes, and later the spleen. Infected leucocytes may be detected in peripheral blood as early as 16 hours after exposure. Continued systemic spread within 3 to 4 days ultimately involves lymph nodes, Peyer's patches, bone marrow, and all parenchymatous organs (Carbrey, Foreign Animal Diseases, 4th Ed. p. 202, U.S. Animal Health Assn., Richmond, Virginia, 1984).

Lesions

Lesions of HC are the direct result of epithelial and endothelial involvement. In acute HC, they are usually characterized by hemorrhage, infarction, and cellular damage. Purplish discoloration of the skin that does not leave the skin upon application of pressure may be observed first. The lymph nodes are the first internal tissues to develop pathologic changes. They may be swollen, edematous, and hemorrhagic in their peripheral areas. Later, the hemorrhage becomes more diffuse, giving affected lymph nodes a mottled appearance. The pharyngeal and submaxillary nodes are usually first affected since they provide drainage from the tonsils. Pigs dying of acute HC generally will develop a variety of lesions in parenchymatous organs. Lesions include splenic infarcts, button ulcers in the large intestine--most frequently in the proximal colon, infarcts in the gall bladder, petechial and ecchymotic hemorrhages in the kidney, a flabby heart, an empty stomach with congestion and hemorrhage in the fundus, catarrhal enteritis in the small intestine, engorged mesenteric blood vessels, and sometimes infarcts and ecchymotic hemorrhages of the lungs. Pigs killed late in the course of chronic HC seldom have the lesions described for acute HC.

In chronic HC, lesions characteristic of secondary bacterial infection are usually observed, such as abscesses in various tissues and occasionally ulcers of the cecum and colon. Chronically infected pigs often die with little or no evidence of hemorrhage. Swine surviving HC more than 30 days will usually have a transverse line of dense bone across the rib shaft, and a general depletion of lymphoid tissue (Stewart, Diseases of Swine, Fifth Ed., Iowa State Univ. Press).

Normal-appearing pigs that died 2 to 11 months after farrowing from carrier sows did not have lesions typical of HC (Van

Oirschot and Terpstra, 1977). The only lesions observed were atrophy of the thymus, and swollen, pale, and moist-appearing mesenteric lymph nodes.

Encephalitis can be visualized on histopathologic examination of the brain and spinal cord of HC-infected pigs. The primary lesions are perivascular cuffing, endothelial proliferation, and microgliosis.

Mummified and stillborn pigs may result from infection with HC virus. The affected fetuses may be edematous and have ascites and deformities of the head and limbs, petechial hemorrhages of the skin and organs, hypoplasia of the lungs and cerebellum, and necrosis of the liver (Stewart, In: Diseases of Swine, 5th Ed., Iowa State Univ. Press, 1981).

Laboratory Diagnosis

In the laboratory, HC may be diagnosed through identification of viral antigens in selected tissues using the fluorescent antibody tissue section test (FATST), or identification of HC virus in infected tissue, using the fluorescent antibody cell culture test (FACCT). Tonsil, pharyngeal or submaxillary lymph node, and spleen are the tissues of choice for HC viral assays. Tonsil biopsies and serum should be submitted if no pigs are available for necropsy. Brain, lung, and kidney should be submitted for differential diagnostic examination.

Results of the FATST of tonsil, lymph node, and spleen can be obtained within 2 to 4 hours of the arrival of the tissues at the laboratory. Hog cholera virus can be isolated within 24 to 72 hours after receipt of infected tissues at the laboratory. If tissues from pigs in the febrile stage of acute HC are received, sufficient virus is usually present for positive FACCT results within 24 hours. However, if convalescing or chronically infected pigs are sampled, 2 to 3 days may be needed to obtain positive results.

The two fluorescent antibody tests are equally sensitive. FATST is performed whenever sufficient tissue is received. With sufficient tissue, the laboratory may also perform differential tests for pseudorabies, parvovirus, enterovirus, transmissible gastroenteritis, African swine fever, salmonellosis, and erysipelas.

Formalized tonsil, brain, pharyngeal lymph node, submaxillary lymph node, and spleen are the tissues of choice for histopathological examination. A tentative diagnosis of HC can be made within a few hours after receipt of formalized brain tissue containing typical vascular changes.

Serum samples can be examined for hog cholera antibodies by the fluorescent antibody neutralization (FAN) test. Results can be obtained within 24 to 48 hours of receipt of sera. Hog cholera infected pigs develop neutralizing antibodies within 14 to 21 days after exposure and commonly have titers of 1:64 to 1:1024 within 5 to 6 weeks (Carbrey, In: Foreign Animal Diseases, p. 202, U.S. An. Health Assn., 1984). Vaccination with MLV HC vaccines will also produce titers of up to 1:256.

With the available HC diagnostic techniques, it is usually not necessary to perform pig inoculation tests. However, pig inoculation must be used to determine the relative virulence of an isolate.

Vaccination

Although hog cholera vaccination is prohibited in the United States, MLV vaccine development has continued in other countries. Highly attenuated HC viruses have been developed. Recent experience with MLV HC vaccines in Mexico has demonstrated that, when the vaccine contains sufficient virus (100 protective units per pig) and is properly administered, they usually protect immunized pigs against fully virulent virus. However, many less attenuated MLV vaccines are still on the market in Latin American countries. Experience with those less attenuated products has shown that healthy pigs are protected against HC, whereas pigs with subclinical salmonellosis have developed acute clinical disease with up to 40 percent mortality.

Where less attenuated MLV vaccines are used, the possibility of creating a carrier sow syndrome exists. If a country is developing a HC eradication program, highly attenuated vaccines could be used to reduce replication and spread of HC virus. However, once clinical disease has been suppressed, vaccination must be stopped to be able to detect remaining pockets of infection and avoid the carrier sow syndrome. Serologic surveillance of slaughter and breeder swine and the progeny of vaccinated swine that are more than 12 to 16 weeks of age, is considered an important component of the final phase of an eradication program. (Dr. G. A. Erickson, National Veterinary Services Laboratories, Ames, Iowa, 515 239-8200).

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*U.S. GOVERNMENT PRINTING OFFICE : 1984 0-460-939/APHI-20065